Cerebral venous sinus thrombosis as presenting feature of Crohn’s disease

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Hepatobiliary and vascular manifestations are rare form of extraintestinal manifestations in Crohn’s disease. We report a 20-year-old man in whom cerebral venous sinus thrombosis was the presenting symptom and preceded the diagnosis of Crohn’s disease. [Indian J Gastroenterol 2004;23;148-149]

Key words: Sinus thrombosis, intracranial

Fig: T1 weighted image of axial section of magnetic resonance image showing hyperintense signal in region of left transverse sinus (arrow)

non-cascating granulomas, consistent with Crohn’s disease. ERCP was normal. The patient refused liver biopsy. He was started on 5-ASA and ursodeoxycholic acid. After 6 months he was asymptomatic; he had no loose motions. Hemoglobin was 11.8 g/dL; serum alkaline phosphatase decreased but was still elevated. He was on anti-epileptic medication as advised by his neurophysician. A final diagnosis of Crohn’s disease with thrombotic complication and probable pericholangitis was made.

Patients with IBD have an increased risk of thrombosis. They tend to suffer thrombosis earlier in life, as in the present case. In a series of 7199 patients with IBD, 92 patients (1.3%) suffered a thrombotic complication. Of these, only 9 patients had cerebral vessel involvement. Hypercoagulable states may arise from active bowel inflammation, factor V Leiden mutation, and defective methylene tetrahydrofolate reductase. However, in more than one half of the patients no predisposing factor can be identified. In our patient, except antiphospholipid antibody and homocysteine level, other investigations were not done.

Cerebral venous sinus thrombosis is more often encountered than arterial thrombosis and usually occurs in a patient with diagnosed Crohn’s disease. In our patient cerebral thrombotic manifestations preceded the diagnosis of IBD by 6 months.

Thrombosis in IBD is a cause for concern because of the high associated mortality and its occurrence in a relatively young population.

References
Enterobiasis mimicking Crohn's disease

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We report a 20-year-old man who presented with abdominal discomfort for 2 months. Colonoscopy showed skip areas with ulceration, resembling Crohn's disease. Biopsies showed chronic inflammation and a non-necrotizing granuloma. An adult pinworm was found in the lumen from an uninvolved segment. The patient responded to mebendazole. [Indian J Gastroenterol 2004; 23:149-150]

Key words: Enterobius vermicularis

Biopsies from uninvolved regions of the colon may provide the diagnosis in patients with patchy colonic disease.

A 20-year-old man presented with history of two months of abdominal discomfort without diarrhea or any other symptom. Colonoscopy showed several superficial ulcerations in the rectum, sigmoid, ascending colon and ileum. The intervening mucosa was apparently spared, giving an appearance suggestive of Crohn's disease. Several biopsies from macroscopically affected zones, as well as from preserved ones, were taken.

Biopsies from the ileum showed an eosinophil-rich lymphoplasmacytic chronic inflammatory infiltrate in the lamina propria. One of the fragments showed an epithelial non-necrotizing sarcoidal-type granuloma; it was coated by a peripheral layer of eosinophils. Giant cells were not observed, and microorganisms could not be found with PAS, Giemsa and Ziehl-Neelsen stains. The colonic mucosa showed eosinophilic colitis. In one of the fragments from a macroscopically non-affected area, an adult Enterobius vermicularis was observed (Fig.). It was lying free in the lumen, and coated by eosinophils. No eggs were found.

A diagnosis of colitis and granulomatous ileitis due to Enterobius vermicularis was established, and the patient was treated with mebendazole. His symptoms responded promptly. He refused follow-up colonoscopy. Four years later, the patient remains free of symptoms.

When endoscopy is used in the diagnosis of inflammatory bowel disease, biopsies should be taken from what appears as normal mucosa. The existence of preserved mucosa between affected areas is important in the differential diagnosis between ulcerative colitis and Crohn's disease.

The present case shows how this feature can also be important in the differential diagnosis between inflammatory and infectious bowel disease. The diagnostic clue in this case was found not in the macroscopically preserved colonic mucosa but in its luminal surface, where the adult worm was noticed. Although granulomas in pinworm infection are not common, they tend to be necrotic when present, which was not so in our patient.

Although fungal infection and tuberculosis are considered as causes of granulomatous ileitis, especially in endemic areas or in immunocompromised patients, pinworms are not so commonly considered. They rarely present with ileal and colonic ulcerative disease at endoscopy. Previous weakness of the mucosa is thought to be necessary for the worm to be able to penetrate it.

Clues to the diagnosis of the disease include endoscopic visualization of the worms and the prominent accompanying eosinophilic inflammatory infiltrate in the lamina propria, as in our case. The latter, although present in some cases of ulcerative colitis, is not a common finding in Crohn's disease. It must be remembered that neither tissue eosinophils nor peripheral eosinophilia is a constant finding in enterobiasis.

In conclusion, we report a patient in whom biopsies from preserved areas of the bowel were relevant in